

In this number

Evaluation is a theme running – in different ways – through the editorials and several articles this month. One editorial board member from The Netherlands discusses the meaning and uses of the “epidemiologic transition theory”. The conclusion seems to be that as a theory it still has some way to go to demonstrate its utility. Another editorial board member from Spain evaluates the experience of his own country in coping with an AIDS epidemic. There are lessons for all European countries here.

Within the field of obstetrics two articles undertake more traditional evaluation: the first of a regionalised multi-tier service in The Netherlands, and the second an overview of the provision of caesarean section in the UK. Not all obstetricians will find themselves willing to accept the

conclusions of either paper, and it is probably true that Cochrane-initiative-acceptable, randomised controlled trials are still needed to make a formal evaluation of these areas of service.

Another five papers in this number relate to various aspects of cardiovascular disease. Three of the papers investigate serum lipids and the other two consider risk factors where intervention is more problematic – gender, socioeconomic status, and geography. The conclusion of the paper from Norway about measuring cardiovascular disease risks is relevant to the evaluation of intervention studies, but for the other four, further work and thought are needed before their relevance to intervention and prevention is clear.

Editorials

The epidemiologic transition theory

The “epidemiologic transition theory” was first formulated in a paper published by Omran in 1971.¹ This theory provides a stylised description and explanation of the mortality component of the “demographic transition”, the spectacular decline firstly of death rates and then of birth rates which has been observed in all currently industrialised countries.² In the epidemiological transition theory, the historical development of mortality over time is characterised by three phases: the “age of pestilence and famine”, the “age of receding pandemics”; and the “age of degenerative and man-made diseases”. It is the transition from a cause of death pattern dominated by infectious diseases with very high mortality, especially at younger ages, to a pattern dominated by chronic diseases and injuries with lower mortality, mostly peaking at older ages, that is seen to be responsible for the tremendous increase in life expectancy.¹ ³⁻⁶ In countries in western Europe and northern America the shift started early and took approximately 100 years. This was called the “western” or “classical model” of the epidemiologic transition. In a number of other countries, notably Japan and eastern Europe, the transition started later but proceeded much more quickly (the so called “accelerated model”). Finally, in many third world countries the transition started even later and, unlike that in currently industrialised countries, has not yet been completed (the “delayed” or “contemporary model”). Omran attributed the decline of mortality to a complex of factors closely linked to “modernisation”. For the western model, socioeconomic progress, leading to a rise in living standards, was presumed to be a very important contributing factor, whereas for the accelerated and delayed models, public health and medical technologies were considered relatively more important.^{1, 3-6}

The concept of an epidemiologic transition (sometimes also referred to as “mortality transition”, or “health transition”) has become popular among demographers and geographers.⁷⁻¹⁰ While it is also well known to many public health professionals,¹¹ it is surprisingly less familiar to

epidemiologists – as is shown by its absence from most epidemiology textbooks and from the International Epidemiological Association’s *Dictionary of Epidemiology*.¹² Perhaps for this reason it has never been subject to the rigorous scrutiny it deserves. In this comment, which will be limited to the “western model” of the epidemiologic transition, I will argue that the concept is ill defined, and cannot therefore be put into operation without ambiguity. The problems come to light rather acutely when one wants to locate in time the beginning and end of the epidemiologic transition.

Should the beginning and end of the epidemiologic transition be based on trends in all cause mortality?

In Omran’s publications, the epidemiologic transition was never clearly defined. His words were the following: “Typically, mortality patterns distinguish three major successive ‘stages’ of the epidemiologic transition: (1) The Age of Pestilence and Famine, when mortality is high and fluctuating (2) The Age of Receding Pandemics, when mortality declines progressively and the rate of decline accelerates as epidemic peaks become less frequent or disappear (3) The Age of Degenerative and Man-Made Diseases, when mortality continues to decline and eventually approaches stability at a relatively low level ...”.¹ If we take this literally, the beginning of the epidemiologic transition lies back in prehistory, because mortality has always been “high and fluctuating”, but it is unclear what changes are supposed to have occurred during the first stage. It seems more appropriate to locate the beginning of the epidemiologic transition *between* the first and second stages, and that of course is what most researchers have done. According to Omran, in most industrialised countries the second stage began “about the middle of the 19th century”.⁴

It is obvious from the citation given above that Omran tended to identify the beginning and end of the transition on the basis of trends in all cause mortality. Given that the whole notion derived from the concept of a demographic

transition, and that the latter is defined in terms of birth and (total) death rates, this may seem a logical step. Recently gained insights, however, raise questions about the appropriateness of this decision. In most countries in western Europe and north America, continuous national mortality series begin somewhere in the 19th century. This leads to serious biases in the assessment of the beginning of the "age of receding pandemics". Recent historical and demographic studies based on other sources have shown that in many western European countries, including England,¹³ France,¹⁴ and Scandinavia,¹⁵ reductions in mortality started long before 1800—possibly in the latter part of the 17th century and more definitely around the middle of the 18th century. One important feature of this early phase of mortality decline was the decreasing amplitude of fluctuations in mortality,¹⁶ associated with the decline of plague, smallpox, and typhus.⁸ Although it is not yet known whether this pre-1800 decline in mortality affected all countries which experienced the western model of the epidemiologic transition, it is clear that at least in a number of countries, and on the basis of all cause mortality trends, the beginning of the epidemiologic transition (that is, of Omran's second stage) should be located much earlier in time than "about the middle of the 19th century".

The end of the epidemiologic transition, although not clearly defined (see the citation given above), should probably be taken as the point in time when mortality rates stabilise after the spectacular decline. It is again difficult, however, to determine this point in time unambiguously. If crude mortality rates are analysed, as in Omran's publications, the picture is distorted by changes in the age composition of the population. More importantly, the pattern was radically different for men and women. In most countries in western Europe and northern America, age standardised mortality rates for women did not stabilise at all, but declined more or less uninterruptedly until the present day. It is only for men that there was a temporary interruption in age standardised mortality decline, which in most countries started in the early 1950s. A renewed decline started around 1970.^{17, 18} This renewed decline has sometimes been referred to as a fourth stage of the epidemiologic transition, the "age of delayed degenerative diseases".¹⁹ The decline of mortality from ischaemic heart disease is one of its main components, although declines in other causes, among these accidents, also make a contribution.¹⁷⁻¹⁹

The question raised by these recent falls in mortality (and by the pre-1850 mortality declines!) is, whether or not they can be covered by the original concept of the epidemiologic transition. Should we include them in this, or are we then stretching the concept too far, and should we perhaps consider distinguishing several epidemiological transitions, just as recent changes in fertility and household patterns have led some demographers to introduce the concept of a "second demographic transition"?²⁰

Should the beginning and end of the epidemiologic transition be based on changes in cause of death patterns?

This question may be answered by defining operationally the epidemiologic transition in terms of changes in cause of death patterns. Unfortunately, the titles of Omran's three stages do not offer much guidance, and are even slightly misleading. Although cholera was a pandemic disease, most of the other infectious diseases that were responsible for a declining mortality, such as tuberculosis, pneumonia, and dysentery, were endemic diseases. Nor is it immediately evident what diseases should be included under the vaguely moralistic heading of "degenerative and man-made diseases".

The main problem with identifying the beginning of the

epidemiologic transition on the basis of changes in cause of death patterns – for example in terms of a declining proportion of deaths due to infectious diseases – is a lack of adequate data. National registration of causes of death in western Europe do not generally date back further than the middle of the 19th century (for example, England and Wales began in 1848²¹), with the exception of the Scandinavian countries (Sweden and Finland began in 1749²²). It is therefore generally impossible to study changes in causes of death from the beginning of the decline in all cause mortality. Another difficulty with the data relates to the classification of causes of death, especially before 1900. The first edition of the *International Classification of Diseases* was introduced in 1900, or shortly afterwards, in many developed countries. Before 1900, each country had its own classification scheme which was based, needless to say, on a body of medical knowledge with which modern epidemiologists are no longer familiar.²³

The problems with identifying the end of the epidemiologic transition are not so much related to the availability of adequate data as to the lack of a valid idea about the causes of death to be included in the analysis. There have been many attempts to find an appropriate label for the group of causes of death which rose in importance during the epidemiologic transition. The proposed labels fall into two families, and show signs of great confusion. The *first* family consists of designations which refer to common aspects of the aetiology, pathogenesis, or prognosis of these conditions. Omran's "degenerative and man-made diseases" is an example, as are the more neutral but non-discriminating "non-communicable diseases and injuries" and "chronic diseases". The latter term is obviously not a good all purpose label because traffic accidents are not included and some infectious diseases may have a protracted course too. "Degenerative and man-made diseases" has an inappropriate undertone of moralism ("man-made"), whereas current views of the pathogenesis of ischaemic heart disease and cancer do not see these as age-related biological processes of "degeneration" any more.

The *second* family of labels consists of designations which refer to the presumed wider causes of the rise of these diseases. Examples are "diseases of affluence", "diseases of civilisation", and "western diseases". Although these terms may seem attractive on the surface, their scientific basis is actually rather weak. Some of the "diseases of affluence", such as ischaemic heart disease, have fallen in later stages of the rise in living standards.²⁴ "Diseases of civilisation" refers to a Euro-centred view of human civilization. "Western diseases" is the most seriously researched concept of the three, but it basically refers to diseases which increase in importance as non-western populations become "westernised" – a process perhaps not unrelated to the changes which occurred in western populations in the past 100–200 years, but not necessarily identical.²⁵ Any selection of causes of death which typically increased in importance during the decline of infectious diseases in western Europe and north America is likely to include ischaemic heart disease, some cancers (lung, breast, pancreas . . .), and traffic accidents,²⁶ but should it also include stroke, peptic ulcer, appendicitis, diabetes mellitus, and suicide, to name but a few more disputable cases? The specific selection of causes to be studied can make a difference for determining the end of the epidemiologic transition, because each of these diseases has its own rise and fall, with large differences in timing.²⁷

Conclusion

The epidemiologic transition theory provides a potentially powerful framework for the study of disease and mortality in populations, especially for the study of historical and international variations. Although its primary purpose was

to describe and explain the spectacular fall in mortality which has occurred in all currently industrialised countries, it can also be used to speculate on the likely consequences of future changes in mortality in countries which are lagging behind those which have already completed the epidemiologic transition: will a fall in infectious disease mortality in currently developing countries lead to a rise in chronic diseases and accidents?²⁸ In addition, this notion of a more or less fixed pattern of changes over time in cause specific mortality may lead us to interpret cross sectional differences between countries in cause specific mortality as being due to a different timing of the epidemiologic transition, which in turn would suggest differences in stage of economic and social development as likely causes.

In order to live up to these expectations, however, it is essential to investigate the historical changes in mortality much more thoroughly than has hitherto been done. Careful reconstruction of national time series of cause specific mortality are a necessary first step,²⁹ and possibly cause specific mortality data from the pre-registration era, which are available for certain subnational populations such as London⁸ and Amsterdam,³⁰ would also be useful. Systematic and comparative descriptive analyses of these time series may disclose common patterns of change, and thereby lead to a clearer notion of the transition (or transitions) which have taken place. Finally, we may try to develop comprehensive hypotheses on the explanation of the epidemiologic transition(s), perhaps incorporating recently accumulated evidence on the link between childhood deprivation and adult heart disease.³¹ Clearly, this enterprise is too exciting to be left to demographers and geographers.

J P MACKENBACH

Department of Public Health
Erasmus University Rotterdam,
The Netherlands.

Member of the JECH Editorial Committee

- 1 Omran AR. The epidemiologic transition; a theory of the epidemiology of population change. *Milbank Memorial Fund Quarterly* 1971;49:509-38.
- 2 Beaver SE. Demographic transition theory reinterpreted. Lexington, MA: Lexington Books, 1975.

- 3 Omran AR. The epidemiologic transition in North Carolina during the last 50 to 90 years: I. The mortality transition II. Changing patterns of disease and causes of death. *NC Med J* 1975;36:23-8 and 83-8.
- 4 Omran AR. A century of epidemiologic transition in the United States. *Prev Med* 1977;6:30-51.
- 5 Omran AR. Epidemiologic transition in the U.S.; the health factor in population change. *Population Bulletin* 1977;32(2):3-42.
- 6 Omran AR. The epidemiologic transition theory; a preliminary update. *J Trop Pediatr* 1983;29:305-316.
- 7 Schofield R, Reher D. The decline of mortality in Europe. In: Schofield R, Reher D, Bideau A eds. *The decline of mortality in Europe*. Oxford: Clarendon Press, 1991.
- 8 Mercer A. *Disease, mortality and population in transition*. Leicester: Leicester University Press, 1990.
- 9 Cleland J, Hill AG. *The health transition: methods and measures. Proceedings of an international workshop, London, June 1989*. Canberra: Health Transition Centre, 1991.
- 10 Phillips DR. *The epidemiologic transition in Hong Kong*. Hongkong: Centre of Asian Studies, 1988.
- 11 Last JM, Wallace RB, Maxcy-Rosenau public health and preventive medicine. 13th ed. East Norwalk CT: Appleton & Lange, 1980.
- 12 Last JM. *A dictionary of epidemiology*. New York: Oxford University Press, 1988.
- 13 Wrigley EA, Schofield RS. *The population history of England 1541-1871; a reconstruction*. Cambridge, MA: Harvard University Press, 1981.
- 14 Blayo Y. Mouvement naturel de la population française de 1740 à 1829. *Population* 1975;30(special issue):15-64.
- 15 Flinn MW. *The European demographic system 1500-1820*. Brighton: Harvester Press, 1981.
- 16 Flinn MW. The stabilization of mortality in pre-industrial Western Europe. *Journal of European Social History*. 1974;3:285-318.
- 17 Thom TJ, Epstein FH, Feldman JJ, Leaverton PE. Trends in total mortality and mortality from heart disease in 26 countries from 1950 to 1978. *Int J Epidemiol* 1985;14:510-20.
- 18 Uemura K, Pisa Z. Trends in cardiovascular disease mortality in industrialized countries since 1950. *World Health Stat Quart* 1988;41:155-178.
- 19 Olshansky SJ, Ault AB. The fourth stage of epidemiologic transition: the age of delayed degenerative diseases. *Milb Quart* 1986;64:355-91.
- 20 Kaa DJ van de. Europe's second demographic transition. *Population Bulletin* 1987;42(1):3-56.
- 21 Logan WPD. Mortality in England and Wales from 1849 to 1947. *Population Studies* 1950;4:132-78.
- 22 Imhof AE, Lindskog BJ. Les causes de mortalité en Suède et en Finlande entre 1749 et 1773. *Annales Economies Sociétés Civilisations* 1974;33:915-933.
- 23 Mackenbach JP. De epidemiologische transitie in Nederland. *Ned Tijdschr Geneesk* 1993;137:132-8.
- 24 Marmot MG, Adelstein AM, Robinson N, et al. Changing social class distribution of heart disease. *BMJ* 1978;2:1109-12.
- 25 Trowell HC, Burkitt DP. *Western diseases - their emergence and prevention*. London: Edward Arnold, 1981.
- 26 Powles J. Changes in disease patterns and related social trends. *Soc Sci Med* 1992;35:377-87.
- 27 Barker DJP. Rise and fall of western diseases. *Nature* 1989;338:371-2.
- 28 Beaglehole R. Cardiovascular disease in developing countries: an epidemic that can be prevented. *BMJ* 1992;305:1170-1.
- 29 Vallin J, Meslé F. *Les causes de décès en France de 1925 à 1978*. Travaux et documents de l'INED cahier no 115. Paris: Presses Universitaires de France, 1988.
- 30 Jansen PC, Meere JMM de, Het sterftepatrioort in Amsterdam 1774-1930. *Tijdschr Soc Geschied* 1982;8:180-223.
- 31 Barker DJP. *Fetal and infant origins of adult disease*. London: British Medical Journal Group, 1992.

AIDS in Spain: lessons learned from a public health disaster

World Health Organisation data currently shows that Spain has reported the highest figures for AIDS in the whole European region. At the beginning of the epidemic, however, the situation was very different, as Spain was one of the countries with the fewest cases.¹ The most prominent epidemiological characteristic of HIV infection in Spain is the great importance of transmission between intravenous drug users (IVDUs), which accounts for 60% of all the AIDS cases reported up to 1993. The Spanish IVDU population could be estimated in 150 000 people; of these 50% were already infected by HIV.²⁻⁴

This dramatic spread could have several causes, and by trying to understand these we may be able to avoid repeating past mistakes we have made as public health professionals or ordinary citizens. In addition, our experience in Spain could be of some value to those countries in which the AIDS problem has not reached this proportion.

A rapid spread of HIV among IVDUs was described in some western cities at the beginning of the epidemic.⁵ Spanish data support this pattern of transmission,⁶ and the HIV prevalence in IVDUs between 1983 and 1985 increased from 11 to 47%. Recently, the incidence rate in Spanish IVDUs was accurately measured and was deemed to be the highest ever reported in western countries - 12 cases per 100 person/years (95%CI 9.6, 14.4) between 1987 and 1992.⁷ The current high prevalence of the infection in Spain makes it even more difficult for risk reduction schemes to succeed. An IVDU who fails to use clean syringes has a higher risk of HIV infection in Valencia (prevalence = 50%) than in Liverpool (prevalence = 3%), to mention one of the European cities with a good record in risk reduction strategies.^{3,8} High prevalence of HIV infection, however, cannot be considered the only predictor of a high incidence. In some Italian cities the